Effects of management and climate on elk brucellosis in the Greater Yellowstone Ecosystem

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Abstract

Every winter, government agencies feed ~6000 tons of hay to elk in the southern Greater Yellowstone Ecosystem (GYE) to limit transmission of *Brucella abortus,* the causative agent of brucellosis, from elk to cattle. Supplemental feeding, however, is likely to increase the transmission of brucellosis in elk, and may be affected by climatic factors, such as snowpack. We assessed these possibilities using snowpack and feeding data from 1952 to 2006 and disease testing data from 1993 to 2006. Brucellosis seroprevalence was strongly correlated with the timing of the feeding season. Longer feeding seasons were associated with higher seroprevalence, but elk population size and density had only minor effects. In other words, the duration of host aggregation and whether it coincided with peak transmission periods was more important than just the host population size. Accurate modeling of disease transmission depends upon incorporating information on how host contact rates fluctuate over time relative to peak transmission periods. We also found that supplemental feeding seasons lasted longer during years with deeper snowpack. Therefore, milder winters and/or management strategies that reduce the length of the feeding season may reduce the seroprevalence of brucellosis in the elk populations of the southern GYE.

Key Words: brucellosis, elk, Yellowstone, disease management, supplemental feeding

Introduction

Supplemental feeding of wildlife can range from private citizens providing pastries to bears (Gray et al. 2004) to government agencies distributing thousands of tons of hay annually to elk around the southern Greater Yellowstone Ecosystem (GYE, Smith 2001). The effects of supplemental feeding on wildlife include altered space-use patterns, survival, reproduction, and densities (Boutin 1990), all of which may also affect disease dynamics (Farnsworth et al. 2005, Rudolph et al. 2006). Using feeding and snowpack data from 1952 to 2006 and disease testing data from 1993-2006, we assessed the relationships between snowpack, supplemental feeding, and brucellosis in elk (*Cervus elaphus*) populations around the southern GYE.

Brucellosis, caused by *Brucella abortus*, is a chronic bacterial disease widespread in many livestock and wildlife populations and is among the most common zoonotic infections worldwide (Godfroid and Kasbohrer 2002, Pappas et al. 2006). Prior to the introduction of pasteurization, dairy products were the primary source of infection in the human population, causing undulant fever, anxiety and depression (Godfroid 2002). *B. abortus* is transmitted within and among wildlife and livestock primarily by contact with infected fetuses and placentas from abortion events (Cheville et al. 1998). *B. abortus*-caused abortions in livestock also result in economic losses and trade restrictions (Thorne 2001, Godfroid 2002).

Brucellosis is particularly contentious in the GYE where elk and bison (*Bison bison*) are among the last reservoirs of infection in the USA (Cheville et al. 1998, Bienen and Tabor 2006). Brucellosis was probably introduced from cattle to bison in the GYE shortly before 1917 (Meagher and Meyer 1994), but due to a successful eradication campaign the cattle populations of most states in the USA are free of the disease (Ragan 2002). To prevent transmission of the disease from bison to cattle, management agencies attempt to restrict bison from leaving

Yellowstone National Park (YNP) and in 2006 over one-fifth of the bison population (>1000) was culled. Despite the intensive management of bison, it was transmission from elk to cattle that caused Wyoming and Idaho cattle to lose their brucellosis-free status in 2004 and 2006, respectively (Galey et al. 2005), costing each state millions of dollars. Wyoming cattle recently regained their brucellosis-free status, but the threat of spill-over from elk and bison remains.

At the center of an elk management debate among environmentalists, ranchers, and managers are 23 supplemental elk feedgrounds maintained by the Wyoming Game and Fish Department (WGFD) and the U.S. Fish and Wildlife Service (USFWS). Supplemental feeding in the southern region of the GYE began in 1910 to limit elk impacts on agricultural land and maintain elk populations despite shrinking native winter range (Smith 2001). Since 1910, elk populations on and off of the feedgrounds have increased dramatically, and in many places around the GYE are above management targets (Dickson 2005). Feedgrounds are intended to minimize contact between elk and cattle during winter, but they also increase the concentration of elk between November and April and the transmission of *Brucella abortus* among elk is most likely between February and June (Roffe et al. 2004). Thus, feedgrounds could sustain or intensify the problems of brucellosis within elk populations, potentially increasing the exposure of neighboring livestock. The average seroprevalence of brucellosis on elk feedgrounds is ~26% while elk populations in other regions of the GYE tend to have a seroprevalence of 2-3% (WGFD unpublished data, Aune et al. 2002, Etter and Drew 2006), and elk outside GYE are not known to sustain the disease.

The elk feedgrounds facilitate a brucellosis vaccination program that began in 1985. Almost all calves are vaccinated annually using Strain 19 biobullets on all feedgrounds except Dell Creek (WGFD unpublished data). In captive studies, the Strain 19 vaccine reduced abortion events from 93% to 71% during the first pregnancy (Roffe et al. 2004). Over the longer term the reduced abortion rate, and thus transmission, may result in lower seroprevalence on the vaccinated feedgrounds. The elk feedgrounds provide a rare opportunity to investigate the ecological and management-related factors driving the prevalence of a chronic disease in a large mammal where sufficient replication is often difficult to attain.

We expect that longer feeding seasons and larger elk populations will be associated higher brucellosis seroprevalence. We first assess how brucellosis status is associated with the date tested, age, elk population size, beginning and ending dates of the feeding season, and the total number of days fed. We then explore the effects of snowpack and proximity to local cattle operations on the timing of supplemental feeding. Elk are typically fed until they leave the feedgrounds as native forage becomes available in spring. Therefore, we hypothesize that length of the feeding season is associated with the variation in snowpack conditions among sites and years.

Methods

Biology of the host and pathogen

Brucella abortus is transmitted among cattle and elk primarily by inducing abortion events or births of non-viable calves. Other individuals are then infected by licking or ingesting the contaminated material (Thorne et al. 1978a). Venereal or airborne transmission of the bacteria is not known to be an important route of infection (Thorne et al. 1978a). Thorne and colleagues (1978a) found that ~50% of infected elk lose their calves in the year following infection; one of nine lost their calves in the second year, and one of five lost a calf in the third year. Live calves born to infected mothers tend to lose their serological titers soon after birth, but some may have latent infections that resurface later in life (Thorne et al. 1978a).

In the GYE, elk comprise the majority of the ungulate community and unfed elk populations tend to aggregate into small herds (13.9 ± 0.67 elk, mean ± SE, Creel et al. 2005), whereas elk that are supplementally fed are in groups of ~260 to 7400 (Appendix A). The supplemental feeding of elk in Wyoming begins between late November and early January, and ends between March and April depending upon the site and year. Data on when abortion events occur are limited, but may range from February to June, whereas natural births occur in May and June (WGFD unpublished data, Roffe et al. 2004).

Supplemental feeding, snowpack and disease testing data

WGFD and the USFWS began recording the beginning and ending dates of the supplemental feeding as well as the number of elk on feedgrounds as early as 1952 with complete data from all feedgrounds from 1980 to 2006. Elk population size was measured annually via a direct census of all individuals on each feedground when peak attendance was expected (usually in January or February). We estimated the maximum elk density at each site by dividing elk population size by the area delineated for typical feeding operations. Feeding area measurements were not available for the National Elk Refuge (NER), so it was excluded from analyses that included density as a predictor.

Disease testing data came from elk captured on four to six feedgrounds per year from January to April using corral traps, helicopter, or ground darting. We excluded tests lacking information on the age of the individual or date of the test. Of the remaining 2136 tests, 55 were tests on individuals that had been captured in previous years. We kept these records in the analyses since this represented less than three percent of all tests. Blood samples were taken from calves, yearling and adult females to determine brucellosis disease status using the following four serological tests: Card test, standard plate agglutination test (SPT), complementfixation test (CF), and Rivanol test. These serological tests indicate whether or not an individual has been exposed, but not whether they are currently infected. We did not include the few samples available on males since they are not known to transmit the infection (Thorne 2001). We interpreted the test results using the U.S. Department of Agriculture (USDA) Uniform Methods and Rules for cervids, whereby reactors were those animals with positive Card tests, Rivanol $\geq$ 1:25 or higher, CF is 2+ at 1:20, and SPT $\geq$1:100 or higher. To differentiate vaccine titers from field strain titers we analyzed samples from 1993 to 2006 using the competitive enzyme-linked immunosorbent assay (cELISA, Van Houten et al. 2003).

Our data on snowpack consisted of snow-water equivalents (SWE; depth of water that would result from melting the snowpack) taken in April from the USDA snowpack telemetry site (SNOTEL) nearest to each feedground (http://www.wcc.nrcs.usda.gov/snow). SWE at nearby SNOTEL sites may not be indicative of local conditions at each feedground, therefore we included elevation of each feedground assuming that this may also be associated with local snowpack conditions. In addition to snowpack data, we used several feedground characteristics as explanatory variables in the analysis of feeding times. Feedground characteristics were taken from a Western Ecosystems Technology Inc. report (2004), which categorized feedgrounds according to the proximity to livestock allotments, whether there had been elk co-mingling issues in the past and the potential for elk damage (Appendix A). The potential for elk damage was ranked on a four point scale, which we collapsed to a binary variable of those considered most at risk versus all other feedgrounds.

Statistical analysis

We used logistic regression models of brucellosis status to assess the role of: beginning (BEGIN) and ending (END) dates of the feeding season; total number of days fed (DAYS); age, testing date (DATE), and elk population size (ELK) and density. Models were ranked according to Akaike Information Criterion (AIC, Burnham and Anderson 2002). Since serological titers may last for several years and due to the lag between exposure and seroconversion (Thorne et al. 1978a, Thorne et al. 1978b) we expected test results to be associated with conditions of previous years. Therefore, we correlated brucellosis status with conditions from the previous year, and the mean over the previous two, four and eight years. Due to the infectious nature of the disease, individuals within a feedground may not be independent of one another. Therefore we included feedground as a variable in all models. We used both generalized linear models (GLM) and generalized linear mixed models with feedground as a fixed and random effect, respectively. The two approaches led to similar conclusions, therefore we present only the GLM results since almost all possible feedgrounds were included in the analyses. We started with a set of 12 *a priori* models, but then expanded upon this set in an attempt to improve upon the best *a priori* model (Table 1). Since many of the feedgrounds were only sampled for one or two years we did not include any interaction terms since many of these parameters could not be estimated.

The inclusion of feedground identity in the models may obscure the effects of other factors because begin and end date as well as population size were more variable across sites than over time. Thus, the inclusion of feedground in the statistical models may confound the effects of other variables. As a result, we used weighted linear regression as an alternative method to assess the relationship between seroprevalence (using all tests at a given feedground) and the begin date, end date, total days fed, and population size and density, which were mean values for each feedground using data from 1990 to 2006. For this analysis we excluded feedgrounds with fewer than 30 serological tests and weighted the other feedgrounds according to the reciprocal of the variance of each estimate (Draper and Smith 1998). Although this method reduced our sample size to 18 feedgrounds and obscured temporal variation it emphasized among site variation and avoided some of the confounding effect of feedground identity. Seroprevalence estimates were not transformed because they did not approach zero or one and the residuals were approximately normally distributed.

The above analyses highlighted the importance of the date feeding ends in the spring (see below). Therefore, our subsequent analyses focused on assessing how snowpack and nearby livestock operations may affect when managers decided to end the feeding season. We first used linear models to assess the correlation between the inter-annual variation in April SWE and end date, averaged across all feedgrounds (1955 – 2006; N = 52 years). We then used a GLM to assess the effects of nearby livestock allotments (presence/absence), elk-cattle co-mingling (yes/no), the likelihood of elk damage to hay stacks (high/low), elevation, and April SWE on the among-site variation in end date, averaged across all years (Appendix A; N = 23 feedgrounds). Finally, we assessed both the spatial and temporal variation in feeding end date using a GLM with all the main effects (END ~ AprilSWE + Damage + Year + ElkPop + Comingle + Livestock + Elevation; N = 903 feedground years). For this analysis we also explored potential interactions among April SWE and elk damage and proximity to livestock allotments but none were found to be significant. We conducted all statistical analyses in R (R Core Development Team 2005).

Results

Both the logistic and linear regression analyses indicated that the supplemental feeding season was strongly associated with brucellosis status and seroprevalence (Fig. 1, Table 1, Appendix B). The weighted linear regression indicated that the length of the feeding season accounted for 58% of the variation in brucellosis seroprevalence among feedgrounds (Fig. 1, β = 0.002 ± 0.0004 SE, p = 0.0008). Both the start and end dates were associated with brucellosis seroprevalence (Fig. 1), however, they were also negatively correlated with one another (r = -0.53, p = 0.018). When we included both start and end date in the same weighted linear regression they became non-significant even though the model predicted brucellosis seroprevalence well (R^2 = 0.59, p = 0.0047, df = 12). As a result, it was difficult to determine from this type of analysis which of these two variables was a more important factor. Due to the influence of the NER, which has low seroprevalence but roughly ten times the number of elk as any other feedground (Appendix A), elk population size was significantly negatively correlated with seroprevalence (data not shown). This result is opposite of what would be expected from theoretical models (McCallum et al. 2001). When NER was excluded from the analysis neither population size nor density were significant (p = 0.66 and 0.77, respectively).

In the logistic regression models individual age, end date, elk population size and feedground were all important factors (Table 1, Appendix B). No calves tested positive (n = 55), 13% of the 478 yearlings were positive, and 26% of 1603 adult females were positive. Although we expected to see more positive test results closer to calving season in the spring, test date did not appear to be an important factor (Table 1). Similar to the linear regression results, the effect of elk population size was negative and heavily influenced by the NER. Although begin date was included in the best *a priori* model, removing begin date resulted in a model that was almost tied for the best *post hoc* model suggesting that begin date may be less important than ending date (Table 1). Further, the parameter estimate on begin date was not significantly different from zero (Appendix B). For the best AIC model, ending date was the most important variable (Table 1, Appendix B). Finally, brucellosis status was predicted best by the average conditions over the previous eight years compared to just the previous year or the mean of the last two or four years (Table 1).

We focused subsequent analyses on those factors that were associated with the end of the feeding season. Feedground identity accounted for 22% of the variation in end date, while inter-annual variation was responsible for 38% of the variation in end date. The inter-annual variation in end date was highly correlated with April SWE, particularly in more recent years (Fig. 2). Site-specific variation in end date was primarily associated with whether or not WGFD personnel perceived the site as having a high likelihood of elk damage to neighboring properties. Feedgrounds where elk damage was more likely were fed on average 10.5 days (95% CI = 4-16) later in Spring than other sites (t-test: $p < 0.003$). The presence of nearby livestock allotments, elevation, and April SWE (from the nearest SNOTEL site) were uncorrelated with site-specific variation in end date, while elk population size was negatively correlated with end date, probably due to the influence of the NER, which had the shortest feeding season (72 ± 5 SE days) but the largest population size (7400 ± 305 SE elk). Finally, we included all factors as main effects into one model in an attempt to explain both the spatial and temporal variation in ending date (Appendix C). This approach showed that feeding seasons have tended to end earlier over time, and feedgrounds with livestock allotments nearby tended to end ~3 days earlier than those that did not (Appendix C). However, the current set of explanatory variables only explained 18% of the total spatial and temporal variation in when managers ended the supplemental feeding season.

Discussion

Many disease models assume that disease transmission is a function of host population size or density (for a review see McCallum et al. 2001). We found, however, that brucellosis was unrelated to the population size and density of elk at each feedground, but was highly correlated with the timing and duration of aggregation. Feedgrounds that continued to feed elk longer had higher brucellosis seroprevalence. Further, the ending date of the feeding season was highly correlated with April snowpack conditions (Figs. 1, 2). Dobson (1996) as well as Joly and Messier (2004) also found only weak or no evidence for a relationship between brucellosis seroprevalence in bison and population size/density. The lack of support for an effect of population size or density may be common to many wildlife disease systems where densities and transmission rates vary seasonally (Altizer et al. 2006). If contact rates are density-dependent then parasite transmission will be proportional to the population density integrated over the time interval of transmission, which for brucellosis is probably limited to the early spring just prior to and during the calving season. Thus, the mixed results of comparative analyses that investigated the effect of population size and density on the immune system or parasite diversity (Cote and Poulin 1995, Nunn et al. 2000, Nunn 2002, Stanko et al. 2002, Tella 2002, Nunn et al. 2003a, Nunn et al. 2003b) may be due to an incomplete understanding of how host population densities fluctuate relative to peak transmission periods.

Data from a captive study of elk suggest that more abortion events occur later in the feeding season from March to June (Roffe et al. 2004). Although begin date was highly correlated with seroprevalence in the weighted linear regression (Fig. 1C), it appeared to be less important in the logistic regression approach (Table 1, Appendix B). Feedgrounds that started earlier also tended to end later. Therefore, some of the importance of begin date may be due to its association with end date. We believe that late-season abortions, and the associated brucellosis transmission events, are the mechanism driving the higher brucellosis seroprevalence on feedgrounds with longer feeding seasons. If the associations shown here reflect causal relationships a 30 day decrease in the length of the feeding season, due to earlier snowmelt or altered management, would result in a drop in brucellosis seroprevalence of approximately two-thirds (Fig. 1C).

The end date of the feeding season was highly variable among sites and years (Figs. 1 and 2). Although feedgrounds with the perceived potential for elk damage were fed longer than other sites, this difference was relatively minor compared to the amount of inter-annual variation associated with snowpack conditions. Feeding seasons lasted up to 30 days longer during years with deep snowpacks and the correlation between the ending date of the feeding season and April SWE has increased over time (Fig. 2). Although future precipitation patterns are difficult to predict, several studies project a future decline in the winter snowpacks of the northern Rocky Mountains (Byrne et al. 1999, Lapp et al. 2002, Lapp et al. 2005, Schindler and Donahue 2006). Our analyses suggest that if the trend over the past 50 years towards earlier snowmelt in the GYE continues (Wilmers and Getz 2005) the feeding season is likely to also shorten, which may result in lower brucellosis prevalence over the long term.

Despite the strong correlation between mean end date each year and April SWE there remains a large amount of unexplained among-site variation in when the feeding season ends. Models including all possible main effects only explained 18% of the total variation (i.e. spatial and temporal) in end date (Appendix C). We hypothesized that the sites and years with more snow would have longer feeding seasons, due to increased nutritional demands of elk. However, the site-to-site variation in the end of the feeding season was unassociated with the snowpack conditions at the nearest SNOTEL site. This suggests three possibilities: 1) SNOTEL sites are not a good indicator of local snowpack conditions at each feedground; 2) other environmental factors, such as hay quality, are more important than snowpack conditions; and/or 3) the end of the feeding season is more related to management decisions than to biological demand. If feeding season length is primarily due to management rather than climate or ecological constraints, then this suggests potential flexibility in feeding season lengths that would allow for experimental manipulation. Experimental manipulation of feeding season length, and in particular the end date of the feeding season, would provide the controlled test necessary to determine if the correlations shown here also reflect causal relationships.

Two feedgrounds in this analysis are particularly noteworthy. The NER feeds more elk than any other feedground (~6700 in 2006 compared to 585±203 SD on other feedgrounds) and of those feedgrounds with over 30 samples it has the lowest brucellosis seroprevalence (seroprevalence = 0.11; 95% CI = [0.07-0.14]). This is possibly due to the short feeding season at NER, which reduces the probability of abortion events later in the season occurring while the elk are on the feedground. The second noteworthy feedground is Dell Creek, which is the only unvaccinated feedground. Nearly all juveniles at other feedgrounds are vaccinated annually with Strain 19 biobullets (Roffe et al. 2004). Previously, the lower seroprevalence on other feedgrounds, compared to Dell Creek, suggested a protective effect of vaccination. Our analyses, however, indicate that the average seroprevalence on Dell Creek is no higher than would be expected given the length of its feeding season (Fig. 1). Thus, the data presented here (though indirect and based on a single population) are not suggestive of a strong protective effect of Strain 19 vaccination at a feedground level, which agrees with previous captive studies (Herriges et al. 1989, Roffe et al. 2004).

This study is the first to identify those factors that explain the variation in brucellosis seroprevalence among the feedgrounds of western Wyoming. Further research is necessary to prove the causality of the relationships found here, but we believe the mechanism of longer feeding seasons facilitating more disease exposure is highly plausible. Decommissioning elk feedgrounds may lead to a decrease in brucellosis seroprevalence among elk in the long-term. Wildlife and livestock managers, however, remain concerned that reduced feeding would lead to increased *B. abortus* transmission from elk to cattle. Whether reduced feeding would result in lower elk population sizes, which may also reduce contact rates between livestock and elk, remains an open question. The management of brucellosis in the GYE is complicated by many political, ecological, and economic factors (Bienen and Tabor 2006), but most constituents have a common goal of maintaining open space and healthy elk populations in one of the fastest developing regions of the United States (Hansen et al. 2002).

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References

Altizer, S., A. Dobson, P. Hosseini, P. Hudson, M. Pascual, and P. Rohani. 2006. Seasonality and the dynamics of infectious diseases. Ecology Letters **9**:467-484.

Aune, K., K. Alt, and T. Lemke. 2002. Managing wildlife habitat to control brucellosis in the Montana portion of the Greater Yellowstone Area. Cheyenne, Wyoming.

Bienen, L., and G. Tabor. 2006. Applying an ecosystem approach to brucellosis control: can an old conflict between wildlife and agriculture be successfully managed? Frontiers in Ecology and the Environment **4**:319-327.

Boutin, S. 1990. Food Supplementation Experiments with Terrestrial Vertebrates - Patterns, Problems, and the Future. Canadian Journal of Zoology-Revue Canadienne De Zoologie **68**:203-220.

Burnham, K. D., and D. R. Anderson. 2002. Model selection and multimodel inference: a practical information-theoretic approach. Second edition. Springer-Verlag New York, Inc., New York.

Byrne, J. M., A. Berg, and I. Townshend. 1999. Linking observed and general circulation model upper air circulation patterns to current and future snow runoff for the Rocky Mountains. Water Resources Research **35**:3793-3802.

Cheville, N. F., D. R. McCullough, and L. R. Paulson. 1998. Brucellosis in the Greater Yellowstone Area. National Academy Press, Washington, D.C.

Cote, I. M., and R. Poulin. 1995. Parasitism and group-size in social animals - a metaanalysis. Behavioral Ecology **6**:159-165.

Creel, S., J. Winnie, B. Maxwell, K. Hamlin, and M. Creel. 2005. Elk alter habitat selection as an antipredator response to wolves. Ecology **86**:3387-3397.

Dickson, T. 2005. Taking aim at depredation. Montana Outdoors **37**:6-7.

Dobson, A., and M. Meagher. 1996. The population dynamics of brucellosis in the Yellowstone National Park. Ecology **77**:1026-1036.

Draper, N. R., and H. Smith. 1998. Applied Regression Analysis. Third edition. Wiley, New York.

Etter, R., and M. L. Drew. 2006. Brucellosis in elk of eastern Idaho. Journal of Wildlife Diseases **42**:271-278.

Farnsworth, M. L., L. L. Wolfe, N. T. Hobbs, K. P. Burnham, E. B. William, D. M. Theobald, M. M. Conner, and M. W. Miller. 2005. Human land use influences chronic wasting disease prevalence in mule deer. Ecological Applications **15**:119-126.

Galey, F., J. Bousman, T. Cleveland, J. Etchpare, R. Hendry, J. Hines, B. Lambert, J. Logan, S. Madden, B. Mead, K. Mills, K. Musgrave, D. Oldham, M. Olsen, T. Pollard, C. Purves, J. Snow, A. Summers, T. Thorne, B. Wharff, and B. Williams. 2005. Wyoming Brucellosis Coordination Team report and recommendations. Cheyenne, WY.

Godfroid, J. 2002. Brucellosis in wildlife. Revue Scientifique et Technique Office International des Epizooties **21**:277-286.

Godfroid, J., and A. Kasbohrer. 2002. Brucellosis in the European Union and Norway at the turn of the twenty-first century. Veterinary Microbiology **90**:135-145.

Gray, R. M., M. R. Vaughab, and S. L. McMullin. 2004. Feeding wild American black bears in Virginia: a survey of Virginia bear hunters, 1998-99. Ursus **15**:188-196.

Hansen, A. J., R. Rasker, B. Maxwell, J. J. Rotella, J. D. Johnson, A. W. Parmenter, L. Langner, W. B. Cohen, R. L. Lawrence, and M. P. V. Kraska. 2002. Ecological causes and consequences of demographic change in the new west. Bioscience **52**:151-162.

Herriges, J. D. J., E. T. Thorne, S. L. Anderson, and H. A. Dawson. 1989. Vaccination of elk in Wyoming with reduced dose strain 19 Brucella: Controlled studies and ballistic implant field trials. Pages 640-655 *in* Proceedings of the U. S. Animal Health Association.

Joly, D. O., and F. Messier. 2004. Factors affecting apparent prevalence of tuberculosis and brucellosis in wood bison. Journal of Animal Ecology **73**:623-631.

Lapp, S., J. Byrne, S. Kienzle, and I. Townshend. 2002. Linking global circulation model synoptics and precipitation for western North America. International Journal of Climatology **22**:1807-1817.

Lapp, S., J. Byrne, I. Townshend, and S. Kienzle. 2005. Climate warming impacts on snowpack accumulation in an alpine watershed. International Journal of Climatology **25**:521-536.

McCallum, H., N. Barlow, and J. Hone. 2001. How should pathogen transmission be modelled? Trends in Ecology and Evolution **16**:295-300.

Meagher, M., and M. E. Meyer. 1994. On the origin of brucellosis in Bison of Yellowstone National Park - a review. Conservation Biology **8**:645-653.

Nunn, C. L. 2002. A comparative study of leukocyte counts and disease risk in primates. Evolution **56**:177-190.

Nunn, C. L., S. Altizer, K. E. Jones, and W. Sechrest. 2003a. Comparative tests of parasite species richness in primates. American Naturalist **162**:597-614.

Nunn, C. L., J. L. Gittleman, and J. Antonovics. 2000. Promiscuity and the primate immune system. Science **290**:1168-1170.

Nunn, C. L., J. L. Gittleman, and J. Antonovics. 2003b. A comparative study of white blood cell counts and disease risk in carnivores. Proceedings of the Royal Society of London Series B-Biological Sciences **270**:347-356.

Pappas, G., P. Papadimitriou, N. Akritidis, L. Christou, and E. V. Tsianos. 2006. The new global map of human brucellosis. Lancet Infectious Diseases **6**:91-99.

R Core Development Team. 2005 R: A Language and Environment for Statistical Computing. Vienna, Austria: R Foundation for Statistical Computing.

Ragan, V. E. 2002. The Animal and Plant Health Inspection Service (APHIS) brucellosis eradication program in the United States. Veterinary Microbiology **90**:11-18.

Roffe, T. J., L. C. Jones, K. Coffin, M. L. Drew, S. J. Sweeney, S. D. Hagius, P. H. Elzer, and D. Davis. 2004. Efficacy of single calfhood vaccination of elk with *Brucella abortus* strain 19. Journal of Wildlife Management **68**:830-836.

Rudolph, B. A., S. J. Riley, G. J. Hickling, B. J. Frawley, M. S. Garner, and S. R. Winterstein. 2006. Regulating hunter baiting for white-tailed deer in Michigan: Biological and social considerations. Wildlife Society Bulletin **34**:314-321.

Schindler, D. W., and W. F. Donahue. 2006. An impending water crisis in Canada's western prairie provinces. Proceedings of the National Academy of Sciences of the United States of America **103**:7210-7216.

Smith, B. L. 2001. Winter feeding of elk in western North America. Journal of Wildlife Management **65**:173-190.

Stanko, M., D. Miklisova, J. G. de Bellocq, and S. Morand. 2002. Mammal density and patterns of ectoparasite species richness and abundance. Oecologia **131**:289-295.

Tella, J. L. 2002. The evolutionary transition to coloniality promotes higher blood parasitism in birds. Journal of Evolutionary Biology **15**:32-41.

Thorne, E. T. 2001. Brucellosis. Pages 372-395 *in* E. S. Williams and I. K. Barker, editors. Infectious diseases of wild mammals. Iowa State Press, Ames.

Thorne, E. T., J. K. Morton, F. M. Blunt, and H. A. Dawson. 1978a. Brucellosis in Elk .2. Clinical Effects and Means of Transmission as Determined through Artificial Infections. Journal of Wildlife Diseases **14**:280-291.

Thorne, E. T., J. K. Morton, and G. M. Thomas. 1978b. Brucellosis in Elk .1. Serologic and Bacteriologic Survey in Wyoming. Journal of Wildlife Diseases **14**:74-81.

Van Houten, C. K., E. L. Belden, T. J. Kreeger, E. S. Williams, W. H. Edwards, E. T. Thorne, W. E. Cook, and K. W. Mills. 2003. Validation of a Brucella abortus competitive enzyme-linked immunosorbent assay for use in Rocky Mountain elk (Cervus elaphus nelsoni). Journal of Wildlife Diseases **39**:316-322.

Western Ecosystems Technology Inc., "Summary of elk feedgrounds operated by the Wyoming Game and Fish Department." (Tech. Rep. National Wildlife Foundation, Missoula, MT, 2004)

Wilmers, C. C., and W. M. Getz. 2005. Gray wolves as climate change buffers in Yellowstone. PLoS Biology **3**:571-576.

Table 1. Selection statistics for logistic regression models of brucellosis status using test results of 2136 female elk from 1993-2006.

Model[1]	df	AIC[2]	ΔAIC[2]
A priori			
Age[3] + Begin + End + Elk + Feedground	23	2067.45	19.5
Age + Begin + End + Elk + TestDate + Feedground	24	2068.99	21.0
Age + Begin + Elk + Feedground	22	2073.77	25.8
Age + End + Elk + Feedground	22	2075.79	27.8
Age + Begin + End + Feedground	22	2083.90	35.9
Age + Begin + Feedground	21	2085.13	37.2
Age + Days + Elk + Feedground	22	2096.19	48.2
Age + Elk + Feedground	21	2101.76	53.8
Age + End + Feedground	21	2102.90	54.9
Age + Days + Feedground	21	2106.64	58.7
Age + Feedground	20	2121.26	73.3
Feedground	18	2189.07	141.1
Post hoc			
Age + Begin + End + Elk + Feedground (previous 8 yr mean)	**23**	**2047.95**	**0.00**
Age + End + Elk + Feedground (previous 8 yr mean)	22	2047.97	0.02
Age + End + Elk + TestDate + Feedground (previous 8 yr mean)	23	2049.97	2.02
Age + Begin + Elk + Feedground (previous 8 yr mean)	22	2096.68	48.7

Age + Begin + End + Elk + Feedground (previous 2 yr mean)	23	2098.69	50.7
Age + Begin + End + Elk + Feedground (previous year)	23	2104.06	56.1

1 Unless otherwise noted begin date, end date, total days fed, elk population size and density were the mean values from the four years prior to the sampling year.

2 AIC: Akaike Information Criterion; $\Delta AIC = AIC_{current} - AIC_{best}$

3 Age was a categorical variable (calf, yearling, adult)

Figure 1. Mean seroprevalence of brucellosis as a function of mean total days fed (A), and the mean beginning (B) and mean ending dates (C) of the feeding season averaged from 1990 to 2006. Both the beginning and ending date are based on the number of days since November 1st. The lines are linear regressions weighted by the reciprocal of the estimated variance in seroprevalence. Point size is proportional to the sample size of serological tests on each feedground.

Figure 2. Effect of April snow-water equivalent (SWE) on the mean feeding end date across all feedgrounds. Black squares represent the years from 1990 to 2006 and grey circles represent the years from 1955 to 1989. The linear regression and associated statistics are based the years from 1990 to 2006.

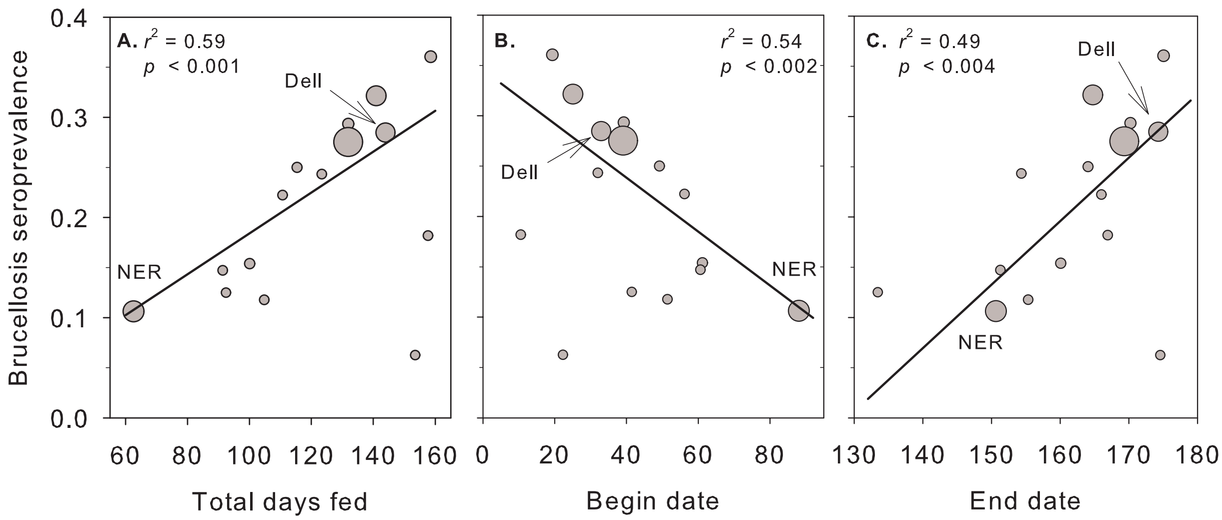
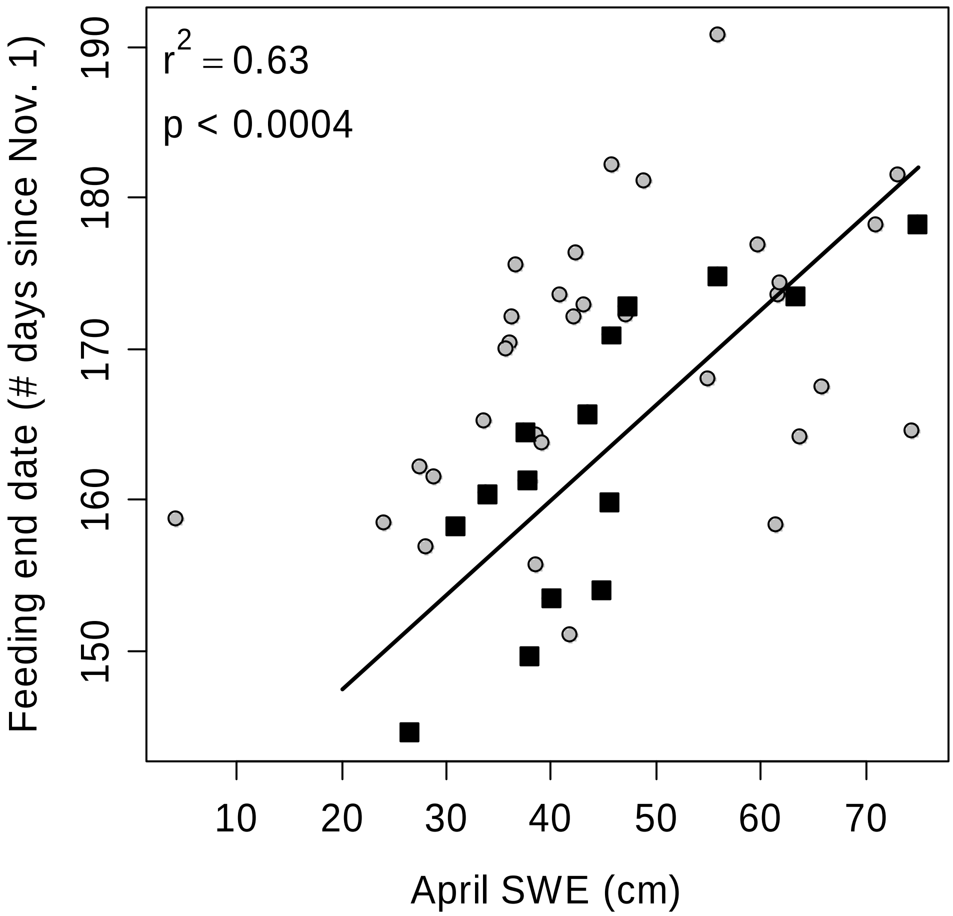

A. r^2 = 0.59
p < 0.001
B.
r^2 = 0.54
p < 0.002
C. r^2 = 0.49
p < 0.004
Dell
NER
Brucellosis seroprevalence
0.0
0.1
0.2
0.3
0.4
60 80 100 120 140 160
0 20 40 60 80
130 140 150 160 170 180
Total days fed
Begin date
End date

Fig. 1



$r^2 = 0.63$
$p < 0.0004$
Feeding end date (# days since Nov. 1)
150
160
170
180
190
10
20
30
40
50
60
70
April SWE (cm)

Fig. 2